

**Dr Paul Champion** said that the case showed clearly the development of hypoxia and dyspnoea. There seemed little doubt that the patient had developed the right-to-left shunt indicated by Dr Keal. Had  $^{133}\text{Xe}$  studies been performed? Dr Keal said he could not prove that the patient did not also suffer from lung fibrosis;  $^{133}\text{Xe}$  studies might help in distinguishing between these two diagnoses.

**Dr J G Lewis** wondered whether smoking an excessive number of cigarettes (around 100 per day) could have any effect on the transfer factor; he had a patient smoking this number daily, who had marked impairment of gas transfer with a normal X-ray and very little evidence of airway obstruction.

### Polycythæmia Masked by Megaloblastic Anæmia

Sylvia M Watkins MRCP

(for Nigel D Compston FRCP)

(Royal Free Hospital, London WC1)

Mr D R, aged 65

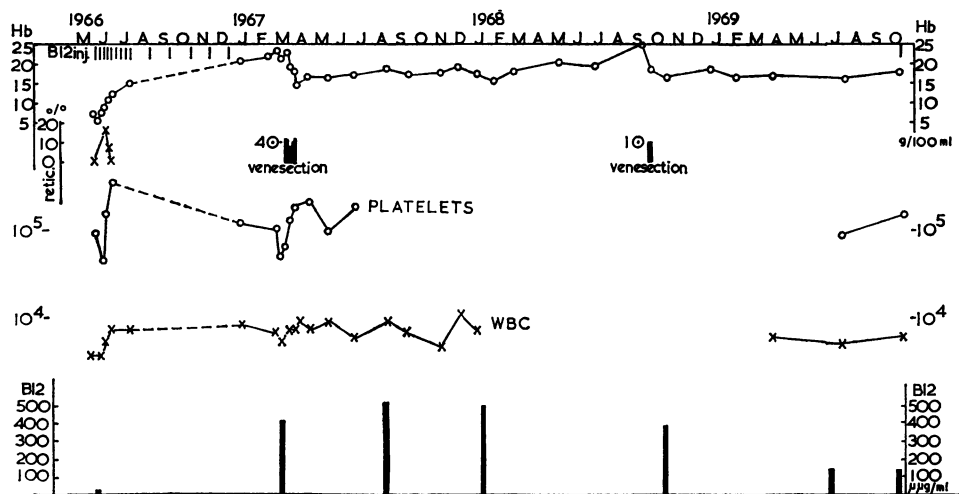
**History:** Has had a duodenal ulcer since the age of 22, when he had a perforation treated by gastrojejunostomy. He continued to have ulcer symptoms, and when he was 46 had a Polya partial gastrectomy. Sixteen years later, in 1966, he complained of tiredness, and was found to have a megaloblastic anæmia: Hb 7.3 g/100 ml; WBC 4,400; platelets 89,000; serum  $\text{B}_{12}$  less than  $10\mu\text{g/ml}$ ; Schilling test part I 2.5%; part II (with intrinsic factor) 20%; histamine-fast achlorhydria; low serum calcium and loss of radiological bone density. He was treated with intramuscular vitamin  $\text{B}_{12}$ , oral iron and calcium. He

had a satisfactory reticulocytosis and the hæmoglobin rose (Fig 1). However, he failed to attend out-patients, and was next seen nine months later, looking plethoric: Hb 23 g/100 ml; WBC 7,700; platelets 48,000–131,000; serum erythropoietin level raised; bone marrow normal; uric acid 8.1 mg/100 ml; arterial oxygen saturation normal; gastric parietal cell antibodies not detected.

### Discussion

At first sight it would seem that this patient had a polycythæmia masked by vitamin  $\text{B}_{12}$  deficiency. Such cases have been described in the literature ever since liver extract was first used in the treatment of pernicious anæmia (PA) (Koessler & Maurer 1927). Birnie (1936) described a patient who oscillated between occlusive vascular phenomena due to polycythæmia, and symptoms of anæmia and subacute combined degeneration of the cord, according to whether or not he received his liver therapy.

Since these early descriptions, over 40 cases of associated PA and polycythæmia rubra vera (PRV) have been published (Galt *et al.* 1952, Hinz 1957, Douglas & Rifkind 1964). Most authors now agree that excess hæmatinics cannot produce polycythæmia, but some believe that there is a relationship between the two conditions (Douglas & Rifkind 1964, Rosner *et al.* 1967). We therefore considered whether our patient might have had true PA, and that his gastrectomy was incidental. He did not have gastric atrophy pre-operatively (stomach histology was normal), and there is no evidence of it now. Klipstein (1962), who reported the only published case of post-gastrectomy vitamin  $\text{B}_{12}$  deficiency followed by PRV, felt that post-operative gastric atrophy may



**Fig 1** Hæmoglobin, reticulocyte count, platelet count (log scale), white cell count (log scale) and serum vitamin  $\text{B}_{12}$  levels from May 1966 to October 1969. Injections of vitamin  $\text{B}_{12}$  (1 mg) are shown at the top of the figure

have been a possibility in his case. England *et al.* (1968) deduced from studies of the incidence of parietal cell antibodies, that there is only a chance association between the two conditions.

The differentiation between primary and secondary polycythæmia was also problematical in this patient. He never had splenomegaly or a raised white cell count, and platelet counts were low; however, none of these findings excludes a diagnosis of PRV. The raised serum erythropoietin suggests secondary erythræmia, but there is no obvious cause for this: he has no cardio-respiratory, renal, endocrine or neurological disease. On the other hand, the raised serum uric acid is suggestive of PRV, which on balance seems the more likely diagnosis.

Therapeutically one cannot withhold vitamin B<sub>12</sub> altogether because of the danger of neuropsychiatric complications. Chalmers & Richards (1961) controlled a patient with PA and PRV on infrequent small doses of vitamin B<sub>12</sub>; however, in spite of low serum B<sub>12</sub> levels, she died of complications of polycythæmia. Alternatively one could give <sup>32</sup>P and B<sub>12</sub> simultaneously: our reason for withholding <sup>32</sup>P was the tendency of this patient to have low platelet levels. Furthermore, there seemed little point in giving him more B<sub>12</sub> when his serum levels were already high. We therefore withheld both drugs, watching his blood count and serum B<sub>12</sub> levels carefully (Fig 1): when the hæmoglobin rose, venesection was performed; and recently, as his serum B<sub>12</sub> level has fallen to the lower limit of normal, injections of B<sub>12</sub> have been restarted.

#### REFERENCES

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#### 'Spontaneous' Left Subphrenic Abscess

J Lerner FRCSed (for J Ian Burn FRCS)  
 (St Charles' Hospital, London W10)

Mr P B, aged 74. Antique dealer

**History:** Emergency admission on 1.6.68 with an irreducible, obstructed inguinal hernia. At operation the hernia was found to contain viable, but congested, loops of small bowel. Herniorrhaphy was carried out without resection. His post-operative course was complicated by temporary ileus which subsided on conservative treatment

and a respiratory infection which responded to antibiotics. Discharged home 11 days after operation.

10.7.68: Readmitted complaining of malaise, rigors and vomiting and a swollen left leg. He was treated with anticoagulants but continued to have fever and rigors with an increasing polymorphonuclear leukocytosis. Repeated X-rays demonstrated a gradual elevation of the left hemidiaphragm with a left pleural effusion. He became anæmic and extremely ill. Repeated blood cultures were negative, as were cultures of fæces. Screening of the diaphragm was initially normal but later showed paradoxical movement on the left. A barium meal (Fig 1) showed separation of the gastric fundus from the left hemidiaphragm (a jejunal and a few large bowel diverticula were also noted). Clinically the spleen became palpable and there was tenderness in the left flank. A diagnosis of left subphrenic abscess was made but during preparations for exploration he suddenly became profoundly ill.

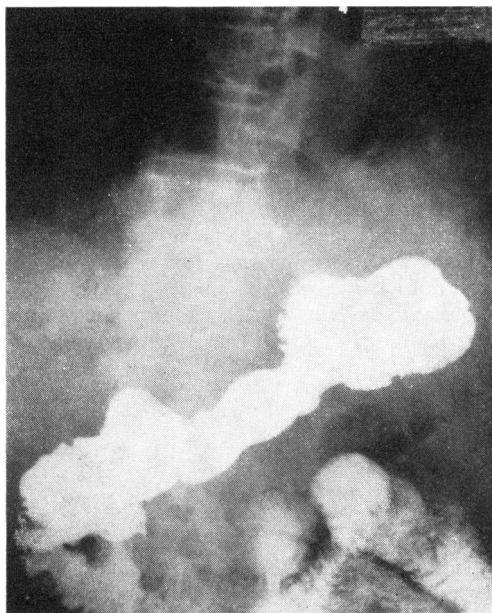


Fig 1 Barium meal demonstrating separation of gastric fundus from left hemidiaphragm

**Operation (27.7.68):** The abdomen was opened through a left upper paramedian incision. The left subphrenic space was distended with pus which had ruptured through the diaphragm into the left thoracic cavity. The spleen appeared normal but was surrounded by omentum. About 500 ml of pus was drained and a tube drain was placed into the abscess cavity. Culture of the pus grew *Salmonella indiana*. There was a past history of 'dysentery' in 1916.